

The Relationship of Attention Deficit Hyperactivity Disorder to Crime and Delinquency: a Meta-Analysis

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ABSTRACT

In recent years, criminologists have begun to focus more closely on how certain biosocial and/or neuropsychological factors may influence criminal and delinquent behaviour. One factor that is

emerging as a potentially important correlate of such behaviour is Attention Deficit — often combined with hyperactivity — Disorder (ADD and/or ADHD). The results of the growing body of empirical literature assessing this link are, however, inconsistent. The present study subjects this body of research to a ‘meta-analysis’ — or, ‘quantitative synthesis’ — to establish both the overall effect of ADHD on crime and delinquency and the degree to which this relationship is conditioned by methodological factors across empirical studies. The analyses reveal a fairly strong association between measures of ADHD and criminal/delinquent behaviour. Nevertheless, these effects are not invariant across certain salient methodological characteristics. The implications for criminological theory and correctional policy are discussed.

Over the course of the past century, 20 different labels were used to describe youths who were inattentive, impulsive, and/or hyperactive — from ‘Fidgety Phils’ to ‘Minimal Brain Dysfunction’ to ‘Hyperkinesis’ (Garber, Garber & Spizman, 1995, p. 5; see also, Conrad and Potter, 2000). It was not until 1980, however, that the American Psychological Association introduced the term ‘attention deficit disorder with or without hyperactivity’ into the *Diagnostic and Statistical Manual* (thus the acronyms of ADD and ADHD). In 1987, the term was officially labelled ‘Attention Deficit Hyperactivity Disorder’. In 1994, the American Psychological Association divided the disorder into three types: a ‘predominantly inattentive type’, ‘predominantly hyperactivity-impulsive type’, and a ‘combined type’ (Garber et al., 1995).

It is estimated that between 3 per cent and 5 per cent of school-aged youths in the United States meet the criteria for ADHD as set forth by the American Psychological Association’s *Diagnostic and Statistical Manual* (Garber et al., 1995; Stern, 2001). Some estimates, however, have placed the range of

youths age 6 to 12 with ADHD from 4 to 12 per cent, with higher estimates for low socio-economic status (SES) youths (Brown et al., 2001; Pineda et al., 1999). The male-female ratio of ADHD cases is believed to be 5 to 1 (Gaub & Carlson, 1997; Stern, 2001). More revealing for our purposes, levels of ADHD among offenders in the criminal justice system may be high. It is estimated that more than a quarter of adult inmates have ADHD (Foley, Carlton & Howell, 1996), and that ‘50 to 80 per cent of prisoners exhibit a significant number of ADHD symptoms’ (Richardson, 2000, p. 18–2; see also, Eyestone & Howell, 1994; McCallon, 2000). Furthermore, ADHD is associated with a variety of conditions that are risk factors for offending, including, for example, neuropsychological deficits, poor academic and cognitive skills, truancy, psychological problems, and defiance and aggression (Barkley, 1998; Brown et al., 2001; Loeber, Stouthamer-Loeber, Van Kammen & Farrington, 1991; Nadder et al., 2001).

Although the precise aetiology of ADHD is not known definitively, the extant evidence suggests that about 80 per cent of the variation in the disorder is genetic/biological. ADHD, for example, is transmitted through biological family lines but not to adopted children (Sprich et al., 2000; Thapar et al., 2000; Weiss, Hechtman & Weiss, 1999). Other risk factors for ADHD include a fetus’s exposure to alcohol and nicotine during pregnancy, obstetric complications, and accidents that affect brain development. Pharmacological interventions — mainly the use of stimulants such as Ritalin — have been found to be effective in treating ADHD (MTA Cooperative Group, 1999). Behavioural therapy, often in combination with medication, also has had success in diminishing the negative behavioural consequences of ADHD (Stern, 2001).

In general, criminologists have ignored ADHD in terms of its theoretical and policy implications. Although exceptions exist (Caspi & Moffitt, 1995), ADHD — or related constructs such as hyperactivity or impulsivity — have been treated most often as one of several risk factors for antisocial/delinquent behaviour (see, eg, Hawkins et al., 1998; Lipsey & Derzon, 1998; Loeber et al., 2001). These assessments are salient, but they do not explore how findings on ADHD might prompt us to reconsider existing theories and to think innovatively about correctional interventions.

In this context, the current study focuses on three issues. First and most important, we endeavour to provide a systematic meta-analysis of the relationship of ADHD to crime and delinquency. Our goal is to establish whether across the extant empirical literature, there is a meaningful association between ADHD and wayward behavior. Second, based on this analysis, we explore the implications of these findings for criminological theory, especially control models. Third, we consider the policy implications of this meta-analysis. Specifically, we examine the implications of the findings for early intervention programmes, for the effectiveness of treatment programmes, and for the management of offender conduct during incarceration.

METHODS

Research strategy

Our current understanding of the empirical status of the major research questions in criminology in general — and of the relationship between ADHD and crime/delinquency in particular — relies almost exclusively on narrative literature reviews (see, eg, Akers, 1997; Burton & Cullen, 1992; Kempf, 1993). While exceptions certainly exist (Gaub & Carlson, 1997; Lipsey & Derzon, 1998; Loeber & Stouthamer-

Loeber, 1986; Paternoster, 1987; Pratt & Cullen, 2000; Tittle, Villemez & Smith, 1978), narrative literature reviews still dominate the criminological landscape.

Even so, in contrast to narrative reviews of research literature, meta-analyses have four potential advantages. First, they are capable of providing more precise estimates of the relationship between certain key theoretical variables and crime/deviance across all empirical tests. Second, they allow the researcher to test for the conditioning effects of various methodological variations on the specified relationships (eg, when variables are measured differently; across different statistical model specifications; in longitudinal versus cross-sectional research designs). Third, since coding decisions are 'public', meta-analyses can be replicated by other researchers. Finally, the database for a meta-analysis is cumulative. As additional studies are conducted and published, they can be added to the sample of studies and the key relationships can be reassessed.

The purpose of the current study, therefore, is to move beyond the narrative review of literature approach to subject the body of empirical examinations of the relationship between ADHD and crime/delinquency to a meta-analysis. In so doing, two issues will be addressed. First, we will calculate the overall mean effect size of ADHD on crime/delinquency, as well as disaggregated mean effect size estimates for the variables specified by the ADHD perspective (ie, where ADHD may be measured differently across studies). This will establish which ADHD variables are the strongest predictors, and also which variables have yet to be adequately tested by researchers. The second issue concerns the degree to which the calculated effect size estimates differ according to methodological variations. To explore this possibility, we statistically examine the potential conditioning effects of certain methodological characteristics —

each of which are discussed below — on the ADHD effect size estimates.

Sample

The sample was generated, in part, by a literature search through a number of electronic databases.¹ Also, consistent with the guidelines set forth by Petrosino (1995), prior narrative reviews of the medical, clinical, and criminological literature were examined to search for additional tests of ADHD and crime/delinquency.² Finally, studies that did not explicitly test the ADHD perspective, but included such measures in their statistical models, were also included in the sample. Overall, the sample includes 20 empirical studies,³ which generated 44 statistical models, containing a total of 48 effect size estimates, representing the integration of 6,261 individual cases.

One potential problem associated with the meta-analysis of empirical literature is that the decision-makers associated with academic journals (ie, editors and reviewers) may be biased in favour of statistically significant findings. Thus, literature searches may not uncover every study of a particular hypothesis that has been conducted. Rosenthal (1979) refers to this as the ‘file drawer’ problem due to the tendency of studies failing to reveal significant relationships to be buried away in file drawers and forgotten. In short, this omission of null-model studies may limit the utility of meta-analyses that are conducted on published research only. As a check on this potential bias, the present study provides a statistical estimate for the number of unmeasured studies that would have to contain a ‘null finding’ to drive the mean effect size estimates calculated here down to zero. In other words, we calculate the number of studies failing to reject the null hypothesis — referred to as the ‘fail-safe N’ — that would be needed to reverse a conclusion

that a statistically significant relationship exists.⁴

Effect size estimate

The effect size estimate used is a standardized correlation coefficient r . This estimate, drawn from each empirical study, was chosen because of its ease of interpretation, and because formulae are available for converting other test statistics (eg, F , t , chi-squared) into an r (see Rosenthal, 1978, 1984). Using Fisher’s r to z transformation (see Wolf, 1986), the effect size estimates from each empirical study were converted to a $z(r)$ score. The correlation coefficients were converted to z -values because the sampling distribution of $z(r)$ -scores is assumed to approach normality, whereas the sampling distribution for r is skewed for all values other than zero (Blalock, 1972).⁵ Normally distributed effect size estimates are necessary for (1) the accurate determination of central tendency for fixed values of the control variables, and (2) unbiased tests of statistical significance (Hanushek & Jackson, 1977).

Separate analyses were conducted on each $z(r)$ after they were weighted for sample size, according to the method recommended by Rosenthal (1984), by taking the product of the $z(r)$ value and the appropriate degrees of freedom (sample size-3) from each study. Weighting the studies on the basis of their sample sizes was done to place a greater emphasis on those studies yielding outcomes from larger samples, which are assumed to be more representative of the population of interest (Rosenthal, 1984; see also Blalock, 1972; Hanushek & Jackson, 1977). Finally, separate analyses were conducted when the mean effect size estimates were adjusted for the potential biases associated with coding multiple effect size estimates from the same dataset (ie, the lack of statistical independence). The correction for this potential bias involved the random effects modelling

approach discussed by Raudenbush and colleagues (Bryk & Raudenbush, 1992; Hedges, 1983; Raudenbush, 1994; Raudenbush, Becker & Kalaian, 1988; see also Pratt, 2000).

Predictor domains

Effect size estimates will first be calculated on the overall effect of ADHD on crime/delinquency with no respect for measurement differences. In essence, as a 'first step' in the analysis we are interested in determining the aggregate effect of these variables — regardless of differences in how they may be measured across studies — on crime and delinquency. We then disaggregate these variables into four separate predictor domains depending on the type of ADHD measure employed in the study. These domains include: (1) *ADD* measures that tap into attention deficits yet exclude a hyperactivity component, (2) *ADHD* measures that combine attention deficits with a hyperactivity component, (3) *hyperactivity* measures that do not incorporate indicators of attention deficit, and (4) some form of *ADHD-combination or index* where concepts such as 'attention problems' (Wallander, 1988) and 'emotional and behavioral problems' (Zahner et al., 1993) are cited as proxies for ADD and/or ADHD. Finally, for each predictor domain, dummy variables are coded as to whether its effect on crime or delinquency was *statistically significant* in the statistical model (0 = no, 1 = yes).

Methodological control variables

Each empirical study was coded for a number of variables related to methodological variations that may condition the effect size of the ADHD predictors on crime and delinquency.

Data source

Two variables were coded from each study indicating the data sources for both the key independent (ADHD) and dependent

(crime/delinquency) variables. The categories included whether the independent and dependent variables were the result of *self-reports*, *official records* (including clinical diagnosis), or the judgment of a *parent or teacher*.

Sample characteristics

A number of factors related to the sample used in an empirical study are coded to assess their impact on the effect size estimates. These characteristics include the sample's *size*, *gender composition* (coded as a categorical variable for mixed gender, males only, or females only), *racial composition* (coded as a categorical variable for racially heterogeneous, whites only, non-whites only, black only, Hispanic only, black and white only), and *age composition* (coded as a categorical variable for juveniles, young adults, adults, or juvenile/adult combination along with a separate variable indicating the mean age of the sample). Additional sampling issues that were coded include the *sampling frame* used by the study (general/community versus school samples), and the *sampling technique* used (coded as a categorical variable for the whole population, a random sample, or a non-random sample). Finally, two control variables were coded regarding the origin of the sample: the first is a categorical variable testing for the *cross-national* applicability of the ADHD-crime/delinquency perspective (with categories for samples drawn from the US, other western nation only, other non-western nation, or a cross-national sample), and the second concerns the *locality* of the sample (local versus national/multi-site sample).

Treatment component

Variables were also constructed to measure the role treatment plays in shaping the relationship between ADHD and crime/delinquency. These variables include whether the *sample had ever gone through treatment* (coded as no/yes), the *nature of the*

treatment (drug treatment, other treatment, or unspecified treatment), and the time of the treatment (past treatment versus current treatment).

Model specification and research design

A number of factors related to each empirical study's model specification and research design were coded to assess their impact on the effect size estimates. Among these factors were whether variables measuring *other personality characteristics* were controlled in the analysis (coded as no/yes) and whether the research design was *cross-sectional* or *longitudinal* (coded dichotomously). Finally, a control variable related directly to the *dependent variable* was coded from each empirical study. The categories include violent crime, property crime, drug use, alcohol use, sexual assault, theft, general crime/delinquency, vandalism, or other.

RESULTS

Table 1 contains the mean effect size estimates for the aggregated and disaggregated ADHD predictors of crime and delinquency. These tables also contain the statistical diagnostics for the variables within each predictor domain — in particular, the mean effect size estimates when weighted for sample size and when adjusted for the potential lack of statistical independence (ie, when multiple effect size estimates were recorded from the same data source). The mean effect size estimates and statistical diagnostics for each of the predictors are discussed first, followed by an analysis of the methodological conditioning effects within each predictor domain and their implications for the interpretation of the overall mean effect size estimates.

Mean effect size estimates and statistical diagnostics

As can be seen in Table 1, there is a statistically significant overall effect of ADHD on

crime and delinquency. Furthermore, the magnitude of the mean effect size estimate for ADHD is large enough ($Mz = 0.155$, $p < 0.01$) to be a 'substantively' important predictor of crime/delinquency (see, eg, the discussion by Andrews & Bonta, 1998; Lipsey, 1992). Nevertheless, there is considerable variation in the mean effect size estimates of the relationships between the various disaggregated ADD and ADHD-related variables and crime and delinquency. These effect size estimates range from the considerably robust predictor of ADD ($Mz = 0.254$, $p < 0.01$), to the more moderate magnitudes of ADHD and hyperactivity ($Mz = 0.081$ and 0.142 , respectively, both are statistically significant at $p < 0.05$), to the weak and statistically insignificant ADHD-hyperactivity combination/index variables ($Mz = 0.078$, $p > 0.05$). In all, indicators of the effect of ADHD-related variables on crime and delinquency tend to be strongest when measured as simply ADD across empirical studies.

Table 1 also contains the statistical diagnostics for each of the predictor domains. Specifically, the mean effect size estimates for each ADHD predictor are calculated when weighted for sample size ($NWMz$) and when adjusted for the potential lack of statistical independence ($RWMz$) from when multiple effect size estimates were recorded from the same data source. In essence, these additional analyses are intended to uncover whether the overall mean effect size estimates (Mz) are significantly biased due to variations in sample size and/or to differential statistical interdependencies.

None of the sample size weighted or random effects weighted mean effect size estimates fell outside of the upper or lower boundaries of the original confidence intervals around the Mz values. Thus, we can be confident that variations in sample size and the potential lack of statistical independence

Table 1: Mean effect size estimates and statistical diagnostics for aggregated ADHD and disaggregated ADD, ADHD, hyperactivity, and ADD-ADHD-hyperactivity combination predictors of crime and delinquency

<i>Variable</i>	<i>Mz</i>	<i>% Sig.</i>	<i>NWMz</i>	<i>RWMz</i>	<i>95% CI</i>
<i>Overall Effects</i>					
ADHD (48)	0.155**	54.2	0.154	0.155	0.135 to 0.175
<i>Disaggregated Effects</i>					
ADD (15)	0.254**	86.7	0.272	0.263	0.119 to 0.389
ADHD (12)	0.081*	33.3	0.079	0.079	0.031 to 0.129
Hyperactivity (16)	0.142*	43.8	0.140	0.137	0.062 to 0.222
ADHD Combo (5)	0.078	40.0	0.027	0.074	-0.030 to 0.186

Note: the numbers of contributing effect size estimates are reported in parenthesis.

Mz = mean effect size estimate.

NWMz = mean effect size estimate weighted for sample size.

RWMz = random effects weighted mean effect size estimate.

* = $p < 0.05$

** = $p < 0.01$

across empirical studies did not significantly bias the overall mean effect size estimates. Also, as discussed above, we conducted an additional diagnostic test to determine the degree to which the mean effect size estimates may be due to the potential existence of a sufficient number of unpublished 'null model' studies — the 'fail-safe N ' statistics. As indicated by Table 1, only the effects of the ADHD combination variables had a fail-safe N less than 20.⁶ Accordingly, for each of the remaining ADHD variables, there does not appear to be a reasonable threat that the mean effect size estimates could be reduced to statistical insignificance by even a substantial number of additional null-model studies.

Methodological conditioning effects

Table 2 presents the series of bivariate analyses assessing the relationships between the various methodological factors and the effect size estimates for each of the disaggregated ADHD predictor domains. These additional analyses reveal four distinct

issues that warrant discussion. First, there is considerable heterogeneity in terms of the degree to which certain types of ADHD variables are conditioned by methodological variations across empirical studies. In particular, the ADHD-combination measures, which also had the weakest mean effect size reported in Table 1, varied consistently and significantly in effect size across most of the coded methodological factors across studies.⁷ Indeed, variables indicating differences in data sources, sample characteristics, and model specification and research designs all significantly conditioned the effect of ADD-combination variables on crime and delinquency.

Second and relatedly, with the exception of the ADD-combination measures the other ADHD variables maintained fairly 'general' effects on crime and delinquency. To be sure, the impact of the methodological variations on the effect size estimates for ADD, ADHD, and hyperactivity variables on crime and delinquency were statistically significant in only eight (14.81 per

Table 2: Impact of methodological variations on estimates of effect size for dis-aggregated ADHD predictors of crime and delinquency

<i>Methodological factor</i>	<i>ADD</i>	<i>ADHD</i>	<i>Hyperactive</i>	<i>ADHD-Combo</i>
<i>Data source</i>				
Independent variable	$F = 1.104$	$F = 0.525$	N/A	$F = 22.472^*$
Dependent variable	$F = 0.460$	$F = 1.301$	$F = 0.002$	N/A
<i>Sample characteristics</i>				
Size	$r = 0.140$	$r = -0.020$	$r = -0.029$	$R = -0.929^*$
Gender composition	$F = 0.182$	$F = 0.992$	$F = 2.216$	$F = 0.128$
Racial composition	$F = 0.285$	$F = 9.033^*$	$F = 0.677$	$F = 22.472^*$
Age composition	$F = 0.297$	$F = 1.781$	$F = 1.573$	$F = 22.472^*$
Mean age of sample	$r = -0.002$	$r = -0.464$	$r = 0.009$	$r = 0.939^*$
Sampling frame	$F = 0.145$	N/A	N/A	N/A
Sampling technique	$F = 0.286$	$F = 1.021$	N/A	$F = 22.472^*$
Cross-national sample	$F = 0.068$	$F = 4.451^\dagger$	$F = 22.135^{**}$	$F = 22.472^*$
Local sample	$F = 0.068$	$F = 0.021$	N/A	$F = 22.472^*$
<i>Treatment component</i>				
Sample gone through Tx	$F = 1.741$	$F = 0.021$	$F = 0.080$	N/A
Nature of Tx	$F = 9.347^\dagger$	$F = 66.124^{**}$	$F = 2.257$	N/A
Time of Tx	$F = 0.360$	$F = 2.453$	N/A	N/A
<i>Model specification and research design</i>				
Other personality variables controlled	$t = -3.15^{**}$	$t = 0.668$	N/A	N/A
Cross-sectional v longitudinal design	$t = -1.647$	$t = 1.188$	$t = -1.261$	N/A
Time lag	$r = -0.479$	$r = 0.973^{**}$	$r = -0.291$	$r = 0.939^*$
Dependent variable	$F = 0.499$	$F = 1.101$	$F = 5.769^*$	N/A

* = $p < 0.05$ ** = $p < 0.01$ † = $p < 0.10$

cent) of the 54 bivariate analyses. A binomial test reveals that this percentage is higher than what could be attributed to random error, yet what may be of more substantive importance is the fact that 85.19 per cent of the bivariate tests for methodological conditioning effects were not statistically significant.

Nevertheless, the third issue revealed by these analyses is that the effect sizes of the ADHD variables on crime and delinquency are not completely invariant across all methodological specifications. Certain

methodological factors did significantly condition the effect size estimates for multiple types of ADHD measures. Specifically, Table 2 indicates that the racial composition of the sample is important, where the effect size of ADHD and ADHD-combination variables are significantly larger in studies where the sample is composed of white subjects. Second, these analyses indicate that samples drawn from western nations other than the United States tend to produce significantly larger ADHD, hyperactivity, and ADHD-combination effect size

estimates. Third, the effect size of ADD and ADHD variables on crime and delinquency are significantly larger among samples that have gone through certain types of treatment (both drug and 'unspecified' treatment).

Finally, Table 2 indicates that the mean effect size estimates of ADHD and ADHD-combination variables are significantly larger in time series designs with a larger time lag. While the time lag variable did not significantly condition the effects of ADD or hyperactivity, the absence of a significant inverse effect for the time lag variable indicates that the effects of ADHD variables on crime and delinquency tend to remain fairly stable over the life course, and, in some instances, it may take on added salience in adulthood.

DISCUSSION

Overall, our study indicates that ADHD is an important risk factor for crime and delinquency. Admittedly, other quantitative reviews of the criminological literature have emerged indicating that other individual-level characteristics may be more strongly associated with wayward behaviour, such as deviant peer associations (Goggin, Gendreau & Gray, 1998), antisocial attitudes (Andrews & Bonta, 1998; Sellers, Pratt, Winfree & Cullen, 2000), and low self-control (Pratt & Cullen, 2000). Furthermore, the effect of ADHD on criminal and delinquent behavior was not invariant across all methodological specifications. Nevertheless, the broader trend in the extant research literature is that ADHD — even when measured differently — tends to have 'general' effects on criminal and delinquent behaviour. To be sure, the effect size is sufficiently substantial as to suggest that the ADHD-crime relationship deserves theoretical explanation and that the

attention-deficit condition warrants consideration in discussions of effective correctional intervention.

Theoretical implications

Beyond the general challenge for theories to account for established empirical relationships, the results of the meta-analysis raise two more specific theoretical issues. First, with the publication of Gottfredson & Hirschi's (1990) 'general theory', the concept of 'low self-control' has emerged as a salient criminological concern. Research has consistently linked low self-control to elevated involvement in delinquency and crime (Pratt & Cullen, 2000). Furthermore, various theories now argue that self-control is a factor that intervenes between theoretically specified causes of criminal involvement. Gottfredson and Hirschi attribute low self-control to ineffective parental management—to the failure of parents to monitor, recognise, and consistently punish deviant conduct. Low self-control is seen by Agnew (1992) as a potential factor that facilitates delinquent coping to general strain, by Colvin (2000) as a 'social-psychological deficit' produced by coercion, and by Cullen & Wright (1997) as fostered by insufficient social support in childhood.

In these theoretical discussions, however, there is a general failure to consider that ADHD is a potential source of low self-control. Although using a limited measure of ADHD, a recent study of over 2,400 middle-school children has, in fact, shown that youths who took medication for attention-hyperactivity problems exhibited lower levels of self-control. These effects were sustained in a series of multivariate models even when controlling for measures of parental management (Unnever, Cullen & Pratt, 2001). The importance of the link between ADHD and self-control is that it suggests that the origins of low self-control

may not be restricted to social experiences (eg, parental management, coercion, social support) but that it also may have genetic/biological origins. As noted previously, although the sources of ADHD are likely multifaceted, a large portion of individual differences in this condition appear to be genetic/biological (Rowe, 2002). If this is indeed the case, then current theories may be incomplete, if not substantially incorrect, in their explanations of the origins of low self-control.

Second, a salient feature of ADHD is that it surfaces in childhood. It is a condition, therefore, that may become entangled with other social and personal risk factors and thus play a role in fostering the early onset of life-course persistent delinquent involvement (see, eg, Moffitt, 1990). One potential impact of ADHD is that it functions as a 'child effect', fostering behaviour that challenges and elicits reactions from parents. Because 'parents and children resemble each other on temperament and personality', notes Moffitt (1993, p. 681), 'parents of children who are difficult to manage often lack the necessary psychological and physical resources to cope constructively with a difficult child'. Instead, they may respond with impulsive, negative reactions — including psychological rejection, verbal abuse, and physical punishment — that create and/or deepen antisocial tendencies. As Moffitt (1990, p. 682) observes, 'it may well be that early behavioural difficulties contribute to the development of persistent antisocial behaviour by evoking responses from the interpersonal social environment . . . [that] exacerbate the child's tendencies' (see also, Caspi & Moffitt, 1995).

In short, to the extent that ADHD is a consistent predictor of youthful misconduct, its role in crime causation warrants further investigation and integration into extant theoretical explanations. Its roles as both a possible source of low self-control

and as a potential catalyst for the early onset of antisocial conduct seem like particularly promising avenues of inquiry.

Policy implications

At least three policy implications can be drawn from the findings of the meta-analysis. First, as noted, an important consideration is that ADHD is a risk factor for crime that emerges in *childhood* and, as a persistent individual difference, tends to have continuing effects over time. The timing of the onset of ADHD is significant, because it means that ADHD is a condition that is a prime candidate for early intervention. It is noteworthy that there is now extensive public support for early intervention programmes (Cullen, Fisher & Applegate, 2000; Cullen, Wright, Brown, Moon, Blankenship & Applegate, 1998). Further, there is growing evidence that early intervention with antisocial children and youths can be effective (see, eg, Currie, 1998; Farrington, 1994; Tremblay & Craig, 1995). In this context, research shows that ADHD is amenable to treatment, with the most favourable results in reducing symptoms and misconduct occurring when children are given multimodal treatment, receiving both medication (in particular) and behavioral intervention (MTA Cooperative Group, 1999; O'Shaughnessy, 1992; Satterfield et al., 1987; Stern, 2001). There is some evidence that youths who manifest ADHD and early antisocial conduct are more resistant to interventions. More success with these kids might be achieved if the interventions not only focused on the ADHD but also specifically targeted for change known predictors of recidivism, such as antisocial attitudes and peer associations (Andrews & Bonta, 1998).

Second, there is a growing literature documenting the 'principles of effective correctional intervention' (Andrews, 1995;

Andrews & Bonta, 1998; Cullen, 2002; Cullen & Gendreau, 2000; Gendreau, 1996). These involve, for example, using cognitive-behavioural programmes, focusing on high-risk cases, and targeting empirically established predictors of recidivism for change. A consideration that has received less theoretical and empirical investigation, however, is the principle of 'responsivity'. This principle suggests that a treatment will be more effective if the intervention takes into account the individual traits of offenders that affect how they 'respond' or react to the style in which the treatment is delivered (eg, degree of programme structure, presence of confrontation). In this regard, ADHD is a personal orientation that is likely to affect how offenders respond to treatments. We might hypothesise, for example, that offenders with ADHD would be more amenable to interventions that are delivered with clear expectations, in a structured format, and in a way that did not require sustained attention (more generally, see Flick, 1998). Regardless, the central insight is that ADHD is commonly found in offender populations and that it may affect treatment effectiveness if it is not taken into account.

Third, as noted, there is growing evidence of a high prevalence rate of ADHD among prison inmates (Comings, 2000; Richardson, 2000; see also, Goldstein, 1997). If so, then it seems likely that this condition is impairing the ability of offenders to cope effectively with the strains and demands of imprisonment (McCallon, 2000). Offenders with ADHD, for example, can be expected to be challenged by impulsivity in an environment that expects conformity, demands from correctional officers to attend to instructions and 'follow orders', and tasks that require lengthy attentiveness (eg, classroom instruction, job assignments). They also may pose problems for other inmates who find their personal styles

aggravating (McCallon, 2000). As McCallon (2000, pp. 17–19) notes, there are 'hidden costs' to misdiagnosing the behaviour of ADHD inmates and of failing to intervene in appropriate ways. For instance, disciplinary infractions might be attributed to wilful disobedience and maladjustment to prison life might be attributed to psychopathology. We are not claiming, of course, that the diagnosis and treatment of ADHD is a panacea for the structural problems inherent in correctional facilities. Still, the failure in the vast majority of prisons to screen for and treat ADHD almost certainly consigns a proportion of offenders to a more painful and less socially productive tenure behind bars.

In summary, the meta-analysis we undertook had the modest goal of assessing whether ADHD could be established, across the existing research literature, as an important predictor of wayward conduct. Again, no claim is being made that ADHD is an enormously powerful cause of crime; rather, the conclusion is that sufficient evidence exists to suggest that it is a meaningful criminogenic risk factor. This conclusion, however, means that theoretical models should consider its effects and explore how ADHD might be integrated into existing explanatory models of crime and delinquency (see, Caspit & Moffitt, 1995). The results also suggest that ADHD is a factor that should be considered in the delivery of treatment services — from early intervention programmes to the rehabilitation and supervision of offenders in adult prisons.

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NOTES

- (1) The electronic databases that were searched include *Articlefirst*, *Criminal Justice*

Periodical Index, Criminal Justice Abstracts, Infotrac, Medline, Psychlit, PsycINFO and Webspirs. Other more general search engines were used as well, including Alta Vista, netscape search, and yahoo search.

- (2) Studies that did not include outcome measures of crime and or delinquency were not included in the sample. For example, certain studies used indicators of 'conduct disorder' as outcome measure being predicted by ADHD, yet others used measures of conduct disorder as proxies of ADHD (Faraone et al., 2000). Thus, to avoid a potential tautology that ADHD researchers themselves acknowledge exists in the extant literature, only those studies predicting either crime and/or delinquency were assessed in the present case.
- (3) These studies include those by Barkley, Fischer, Edelbrock & Smallish (1990), Crowley et al. (1998), Disney, Elkins, McGue & Iacono (1999), Fergusson, Horwood & Lynskey (1993), Fergusson, Lynskey & Horwood (1997), Halikas, Meller, Morse & Lyttle (1990), Hechtman, Weiss & Perlman (1984), Manuzza, Klein, Konig & Giampino (1989), Moffitt (1990), Roizen et al. (1996), Satterfield, Hoppe & Schell (1982), Satterfield, Satterfield & Schell (1987), Satterfield & Schell (1997), Satterfield, Swanson, Schell & Lee (1994), Tarter, Hegedus & Gavalier (1985), Thompson, Riggs, Mikulich & Crowley (1996), Virkkunen & Nuutila (1976), Wallander (1988), Windle (1993), Zahner, Jacobs, Freeman & Trainor (1992).
- (4) This statistic, the 'fail-safe N' (Rosenthal, 1979; see also Wolf, 1986), is calculated using a 0.05 significance level by the formula:

$$N = (\sum z\text{-scores} / 1.645)^2 - N(\text{number of contributing effect size estimates})$$

- (5) The equation for the transformation of r values to $z(r)$ values (see Blalock, 1972), which converts the sampling distribution of r to one that approaches normality is:

$$z(r) = 1.151 \log[1 + r / 1 - r]$$

- (6) One of the problems facing the fail-safe N statistic is that appears to be most informative when synthesising an extremely small number of studies (fewer than 10; see Wolf, 1986). Indeed, with the exception of the ADD-combination variables, in the present case the fail-safe N estimates were consistently higher than 50 (even when the 0.05 benchmark was reduced to 0.001). Thus, the fail-safe N estimates were generally too large to be substantively informative. Hence, the standard of 20, while somewhat arbitrary, was established to help illustrate where a mean effect size estimate could conceivably be 'washed out' with the publication of a *reasonable* amount of null model studies.
- (7) Chi-squared tests for heterogeneity revealed the existence of an outlier in the distribution of effect sizes for the ADHD-combination measures. Furthermore, given the small number of contributing effect size estimates in this predictor domain ($n = 5$), the instability in the estimates of effect size for these variables is not necessarily unexpected.

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